

A STUDY ON CLINICAL PROFILE OF RIGHT VENTRICULAR MYOCARDIAL INFARCTION

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CERTIFICATE

This is to certify that this dissertation entitled “**A STUDY ON CLINICAL PROFILE OF RIGHT VENTRICULAR MYOCARDIAL INFARCTION**” submitted by **Dr. DHANASEKARAN. S** appearing for Part II M.D. Branch I General Medicine Degree examination in March 2010 is a bonafide record of work done by him under my direct guidance and supervision in partial fulfillment of regulations of Tamil Nadu Dr. M.G.R. Medical University, Chennai. I forward this to Tamil Nadu Dr.M.G.R. Medical University, Chennai, Tamil Nadu, India.

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INTRODUCTION

Acute Myocardial Infarction remains among the most common problem encountered in Internal Medicine and Cardiology.

Right Ventricular Myocardial Infarction is different from that of the left ventricle in the acute presentation, therapy and long term prognosis. The early recognition of Right Ventricular Myocardial Infarction is important, because the time of onset of its haemodynamic consequence is unpredictable and these may be prevented by the administration of intravenous fluid load.

Isolated Right Ventricular Myocardial Infarction is very rare. It is usually associated with Inferior Wall Myocardial Infarction and is responsible for the occurrence of bradycardia and heart blocks. It is less commonly associated with Anteroseptal Myocardial Infarction.

The incidence of Right Ventricular Myocardial Infarction in Inferior wall infarction varies from 25-52 % in various studies³⁻¹⁰.

Involvement of right ventricle is related to severe atherosclerotic occlusion of the right coronary artery and is associated with involvement of postero inferior wall and posterior portion of septum.

Clinically Right Ventricular Myocardial Infarction can be suspected when a patient with Inferior Wall Myocardial Infarction presents with elevated JVP, positive Kussmaul's sign, hypotension, right sided third or fourth heart sounds, tender hepatomegaly, oliguria rarely TR and clear chest.

There are various electrocardiographic criteria to diagnose Right Ventricular Myocardial Infarction^{11,12,13}. Chou et al¹⁴ proposed that ST elevation in V1 might suggest Right Ventricular Myocardial Infarction. Candell¹⁵ et al described correlation between ST elevation in V4R and hemodynamic and scintigraphic findings for Right Ventricular Myocardial Infarction.

Introduction of hemodynamic studies, radionuclide ventriculography, pyrophosphate scintigraphy, phonocardiography and 2D echocardiography have made the diagnosis of subclinical cases easier and earlier^{16,17,18}.

AIM

- To study the various Clinical manifestation of Right Ventricular Myocardial Infarction.
- To study the Clinical features, Risk factors, ECG features, Complications and to evaluate the Outcome of Right Ventricular Myocardial Infarction.

REVIEW OF LITERATURE

Historical Aspects

The term 'anginal pain' was first described by Herberden¹⁹ in 1768.

Right Ventricular Myocardial Infarction (RVMI) was first reported as a separate clinico pathological entity in 1930 by Sandas. In their review of 2000 consecutive necropsies in 1948, Wartman and Hellerstein¹ described 22 instances of Right Ventricular Myocardial Infarction out of 164 cases of acute Myocardial Infarction. Wade et al² noted at necropsy, the major damage in Right Ventricular Myocardial Infarction was to the inferior wall.

Right Ventricular Myocardial Infarction was described as a separate clinical entity by Cohn et al³ in 1974.

The electrocardiographic studies on human heart, was first introduced by Waller²⁰ in 1887. Einthoven²¹ elaborated the electrocardiographic study and published Einthoven's law of triangle in 1912. Wilson et al²² introduced unipolar limb and chest leads in ECG. The diagnosis of Right Ventricular Myocardial Infarction became practical and easy when Erhardt et al⁴ introduced right precordial electrocardiography.

Modern era said to have begun with the autopsy studies of Herrick²³ in the year 1912. He concluded that the clinical syndrome of Myocardial Infarction result from acute thrombotic occlusion of coronary arteries, with resulting downstream necrosis.

Right Ventricular Myocardial Infarction was identified as a unique clinico pathological and hemodynamic entity by Cohn³ and his associates in 1974. This ushered a new era in the diagnosis and management of Right Ventricular Myocardial Infarction.

Coronary Circulation

The heart is supplied by two coronary arteries namely the left and right coronary artery. Left main coronary artery arises from the left coronary cusp and divides into left anterior descending coronary artery and left circumflex coronary artery.

Right coronary artery arises from the right coronary cusp of aorta and passes forward and to the right to emerge between the root of the pulmonary trunk and right atrial appendage. It then descends in the right atrio-ventricular groove, curves posteriorly at the acute margin of the right ventricle to reach as far as the posterior interventricular groove. It ends by anastomosing with left circumflex artery.

Right coronary artery gives the posterior interventricular branch (or) posterior descending branch which runs along the posterior interventricular groove and ends by anastomosing with left anterior descending artery near the apex of the heart.

Right coronary artery supplies the posterior one third of the interventricular septum and the adjacent part of both ventricles. It also supplies A-V node, proximal His bundle and posteromedial papillary muscle. The right coronary artery also gives many branches to supply the free right ventricular wall. It supplies the sinus node in 60% of population.

So, if there is a proximal obstruction of right coronary artery there is infarction of right ventricle along with infarction of the inferior wall of the left ventricle, whereas if the obstruction is distal to the branches supplying right ventricle, there is infarction of inferior wall of left ventricle only.

Variations:

1. The two coronary arteries may arise separately (or) from the same sinus.
2. Sometimes 3 or 4 coronary arteries may be present.
3. Right coronary branch, conus artery which is usually its first branch (64%) but sometimes arises separately in the anterior sinus as a third coronary artery.

4. Left coronary opening may be double.

- ❖ Right dominance means the posterior interventricular artery arise from right coronary artery 90%.

- ❖ Left dominance means the posterior interventricular artery arise from left coronary artery 10%.

In chronic coronary obstruction and ischemia, collateral connection develops via subendocardial, subepicardial, transseptal and transatrial vascular connections and the vascular beds become variably interdependent.

Epidemiology

Acute Myocardial Infarction continues to be a major public health problem in both developed and developing countries, despite impressive strides in the diagnosis and management.

Although acute myocardial infarction death rate has declined by about 30% over last decade, it is still a fatal event in approximately one third of patients.

In our country

- ❖ While coronary artery disease had been halved in west in the past 50 years, the rates have been doubled in India²⁴.

- ❖ Coronary artery disease prevalence in Urban areas–9.67%.

Rural areas – 3.7 %²⁵⁻²⁷

- ❖ Extreme prematurity, coronary artery disease mortality in an Indian <30 years of age is 3 fold higher than whites²⁸
- ❖ Women have rates similar to men despite smoking being uncommon in Indian postmenopausal women²⁹

Although atherosclerosis is the most common cause of luminal narrowing in coronary artery disease, multiple non-atherosclerotic causes account for 4-7% cases of acute myocardial infarction^{31,32}

Pathogenesis and Pathology

Coronary atherosclerosis in the form of stenosing plaques is the major etiological factor behind the various clinical syndromes of ischemic heart disease. In a small minority, non atherosclerotic causes like emboli, vasculitis and dissection may be seen.

Stenosis caused by atheromatous plaque may be eccentric or concentric³³. Eccentric plaque causes total variation in lumen size leading to variable luminal stenosis. Concentric plaque has fixed stenosis³⁴.

Fatty streaks - which are the earliest lesion in atherosclerosis starts in coronaries of children, by 10 years. With increasing age, fatty streaks evolve into fibromuscular plaques³⁵.

Platelet adherence, platelet aggregation and release of smooth muscle growth factors cause the embryonic atherosclerotic plaque to increase in their size.

Smooth muscle and fibrous tissue growth factors are released from disturbed endothelium and macrophages. Abnormal lipid transport from arterial lumen followed by necrosis and calcification occurs in atherosclerotic plaque.

Infarction occurs when an atherosclerotic plaque fissures, rupture or ulcerate and when condition favour thrombogenesis, so that mural thrombus forms at the site of rupture and lead to coronary occlusion.

Atherosclerotic plaque can be classified according to morphology into stable and unstable plaque³⁶.

Mechanism of thrombosis

Three forms of vascular injury were proposed by Ip et al³⁷

Type I - Endothelial dysfunction.

Type II - Endothelial denudation and intimal damage with intact

internal elastic lamina.

Type III - Endothelial denudation and damage of both intima and media.

Rupture of plaque is a function of internal plaque changes and external stress.

Internal – Plaque vulnerability

External – Systemic triggers – Blood pressure changes,

- Cigarette smoking, Cold exposure
- Exercise, emotions, erect posture
- Local triggers – Infection, Inflammation³⁴

Platelet activation with release of thromboxane A₂ is an accompanying phenomenon – Hirsch et al.³⁸

It promotes conformational change in glycoprotein II b/ III a receptor leading to platelet aggregation. Then the coagulation cascade activated leading to conversion of prothrombin to thrombin which then converts fibrinogen to fibrin. Ultimately coronary artery becomes occluded by a thrombus containing platelet aggregates and fibrin strands.

Risk factors for Atherosclerosis / Acute Myocardial Infarction³⁹

Category – I

Non modifiable risk factors

- Age
- Male gender

- Family history of early onset coronary artery disease
- Low socio economic status

Modifiable risk factors

- ↑ Low density lipoprotein
- ↓ High density lipoproteins
- Atherogenic diet
- Cigarette smoking
- Hypertension
- Left ventricular Hypertrophy
- Thrombogenic factor

Category II

- Diabetes mellitus
- Physical inactivity
- Elevated triglycerides
- Obesity

Category III

- Lipoprotein(a)
- Homocysteine
- Inflammatory markers - particularly C reactive protein
- Postmenopausal status

In India⁴⁰

- High prevalence of diabetes , insulin resistance syndrome and central obesity
- Emerging newer risk factors like high lipoprotein (a) homocysteine, apo B, triglycerides, fibrinogen, platelet activating factor inhibitor - I
- Low levels of high density lipoprotein and high density lipoprotein 2b
- Small dense low density lipoprotein

Circadian variation

Acute Myocardial Infarction has marked circadian periodicity with peak prevalence between 6 A.M. and noon. There is 3 fold increase in frequency of infarction at around 9 A.M.⁴¹. This is due to diurnal variation in thrombotic tendencies like platelet aggregability⁴², circadian variation in blood pressure⁴³ and sympathetic nervous system⁴⁴.

Right Ventricular Infarctions are less common – why?

Right ventricular myocardial infarction occurs less commonly than would be expected from the frequency of atherosclerotic lesions involving the right coronary artery. Various reasons have been offered to explain this disparity^{9,45-50}.

- a. The thinness of right ventricular wall allows some nutrition to be derived directly from the blood within right ventricular cavity.
- b. The intercoronary collateral system of the right ventricle is richer than that of the left ventricle.
- c. Oxygen demand of the right ventricle is low.
- d. Some parts of the right ventricle are directly nourished by thebesian vessels.
- e. Collateral flow to the right ventricle is both systolic and diastolic, whereas it is mainly diastolic for the left ventricle.
- f. Energy requirement per gram of functioning myocardium is lower for the right ventricle. So the same degree of ischemia is better tolerated by the right ventricle without getting infarcted.

Collateral circulation

An important collateral to the posterior circulation is via the Kugel's arteria anastomotica. It may arise from the proximal right coronary artery or the left main coronary artery and runs from anterior to posterior along the base of the atrial septum. Helen, Grover et al⁵¹ have suggested that moderator band

artery (which is upto 1 mm in diameter) may provide a major source of collateral flow through the anterior papillary muscle of the right ventricle.

Wade² emphasized that in cases of right coronary occlusion, right ventricle receives much of its blood supply through the left coronary artery, particularly through branches that traverse the septum and anterior one third of right ventricle. He claimed that, though right ventricular infarction occurs as a result of right coronary occlusion, preexisting lesions in the left anterior descending artery plays an important role in the pathogenesis.

The anastomosis between the fine branches of right coronary artery are poorly developed at birth and they progressively increase as age advances. This involves the newly formed fine vessels in the epicardial fat (rami telae adipose). However these are not usually adequate enough to maintain an efficient collateral circulation if coronary artery branch is obstructed.

Isolated Right Ventricular Infarction

Isolated right ventricular infarction is very rare and has been reported in the recent past in a few studies.⁵²⁻⁵⁷ In the most cases it was

due to the occlusion of a right marginal artery (or) a small right coronary artery which vascularise only the right ventricle.

Colantorio et al⁴⁷ have postulated that in a few of their cases, right ventricular infarction might have been secondary to acute or chronic pulmonary hypertension especially in the presence of right ventricular hypertrophy with normal or stenotic coronary arteries.

Right atrial infarction was noticed along with right ventricular infarction in 14-35% of cases. The greater incidence of right atrial involvement is explained by the fact that right coronary artery is the most important source of blood to the right atrium.

Sometimes isolated right ventricular myocardial infarction may mimic additional inferoposterior left ventricular infarction on surface electrocardiography as reported by Mittal⁵² et al. This probably might have been due to a dilated right ventricle.

Hemodynamic Changes in RVTI

The hemodynamic changes observed in right ventricular myocardial infarction are secondary to right ventricular dysfunction. The salient features include

- a) Right sided filling pressure greater than left sided filling pressure
- b) An inspiratory increase in mean right atrial pressure and right ventricular end diastolic pressure
- c) Normal or mildly increased left ventricular filling pressure
- d) Normal systolic pressure in the right ventricle and pulmonary artery

The hemodynamic criteria required to diagnose right ventricular myocardial infarction is that the right atrial (or) right ventricular end diastolic pressure must be equal to or greater than a simultaneously measured left ventricular end diastolic pressure or pulmonary capillary wedge pressure, provided the right atrial pressure is > 10 mm Hg. If right atrial pressure is less than 10 mm Hg⁵⁸, a volume loading test is done following which the pressures are measured again.

These changes result from decrease in the compliance of right ventricle and possibly also due to the resistance offered by the pericardium to the dilating heart. A severe non compliant pattern of right ventricle is defined as Y descent deeper than X descent in right atrial pressure tracing. These changes are

responsible for right ventricular S₃, S₄. Right ventricular infarct may mimic constrictive pericarditis, when there is equilibration of the diastolic pressure of both sides, a diastolic dip and plateau is present in both right and left pressure tracing and there is no inspiratory fall in the right atrial pressure. These hemodynamic features of right ventricular myocardial infarction may not be seen in the presence of hypovolemia.

Low output Syndrome in RVMI

The low output syndrome in right ventricular myocardial infarction is attributed to inadequate left ventricular filling. When right ventricular contractions are impaired severely as in right ventricular myocardial infarction, the filling of the left ventricle is entirely dependent on the distension of the right ventricle and right atrium by any of the following mechanisms⁵⁹.

- a) An increase in right ventricular end diastolic volume which could augment the contractile force of the residual functioning fibres of the right ventricle
- b) Increased right atrial contractile force which might help to pump the blood into the pulmonary artery in the absence of right ventricular contraction

c) Increased passive blood flow across the right to left atrial pressure gradient, through the low resistance pulmonary vascular bed.

Right ventricular ischemia increases the left ventricular chamber stiffness with shift of the interventricular septum to the left, which is enhanced by the restrictive effects of the pericardium. This also contributes as to the low output state in right ventricular myocardial infarction.

History, Physical examination and Management of Acute Myocardial Infarction

The classical symptom of acute myocardial infarction is Chest discomfort which is retrosternal or pericardial described as pressure, aching, burning, crushing, squeezing, heaviness, sweating or bursting in quality. It is usually radiated to medial aspect of arm, neck or jaw, back etc⁶⁰.

Approximately 23 % of acute myocardial infarction has the following atypical clinical features

- ❖ Nausea or vomiting/ indigestion (common with inferior wall myocardial infarction)
- ❖ Atypical location of pain-arm, back , jaw, occiput

- ❖ Profound fatigue of rapid onset
- ❖ Pulmonary edema of sudden onset
- ❖ Mental obtundation
- ❖ Severe ventricular dysrhythmias
- ❖ Confusion, vertigo, syncope
- ❖ Cerebral or peripheral embolus

The older the patient, the more atypical symptoms manifest⁶¹. Painless myocardial infarction is common in elderly and diabetics due to autonomic dysfunction.

Examination - features of Inferior Myocardial Infarction

General Examination

- ❖ Patient is anxious and restless
- ❖ Pallor with respiration and coolness of the extremities
- ❖ Temperature elevation upto 38° celcius during the first week

Vital Signs

The acute inferior wall myocardial infarction is very often associated with transient hypotension and sinus bradycardia due to Bezold Jarisch reflex. This is the reflex response of bradycardia and hypotension due to the stimulation of

inhibitory cardiac receptors in the infero posterior wall of left ventricle. About half of inferior wall infarction shows evidence of parasympathetic hyperactivity

Rhythm of pulse

Inferior wall myocardial infarction frequently produces sinus node and AV node dysfunction manifested as irregularities in pulse.

Pulsus paradoxus occurs if right ventricular myocardial infarction complicates inferior wall myocardial infarction

JVP

Right ventricular involvement in inferior wall myocardial infarction is associated with JVP changes

- Elevated JVP
- Prominent 'a' waves
- Kussmaul's sign

AV dissociation associated with inferior wall myocardial infarction produces cannon 'a' waves.

Precordial Palpation

- Precordium is usually quiet
- Diffuse apical impulse

- Dyskinesias – paradoxical bulge in late systole
- Palpable S₄
- Paradoxical splitting rarely due to severely left ventricular dysfunction

Auscultation

- Muffled heart sounds
- Muffled S₁ common in acute inferior wall myocardial infarction due to prolonged PR interval
- S₄ - 98% cases of acute myocardial infarction⁶²
- S₃ - 15-20 % cases of acute myocardial infarction⁶³
- Pericardial rub
- Crescendo – decrescendo mid systolic murmur due to ischemia of papillary muscles
- Mitral regurgitation murmur
- Pan systolic murmur due to Ventricular Septal rupture

Examination of Lung fields :

Pulmonary congestion occurs in 30-40% cases of uncomplicated myocardial infarction

Killip's Classification

- Class – I : No pulmonary rales or S₃
- Class – II : Bibasilar rales that persist after coughing (< 50 % lung fields) or S₃
- Class – III : Rales over one - half of the lung fields bilaterally with radiographic evidence for pulmonary edema (> 50 % of lung fields)
- Class – IV : Cardiogenic Shock

Clinical features of Right Ventricular

Myocardial Infarctions

Patients with right ventricular myocardial infarction will show clinical features of right ventricular dysfunction only when the infarction is haemodynamically significant

Salient clinical features of a dominant right ventricular myocardial infarction can be summarised as below.

Symptoms - The patient presents with retrosternal chest pain associated with sweating, nausea and vomiting. Giddiness and syncopal attacks may be present. Dyspnoea is usually uncommon.

Signs - Systemic hypotension will be present often associated with peripheral vasoconstriction. Pulsus alternans and pulsus paradoxus may be present depending on the severity of the right ventricular myocardial infarction. Mean JVP is usually elevated and Kussmaul's sign may be present. Patient may have oliguria. Auscultation of the heart may reveal a right ventricular S_3 , S_4 and a pansystolic murmur of tricuspid regurgitation. Lungs are usually clear without any evidence of pulmonary vascular congestion. Liver may be palpable and tender.

Kussmaul's Sign – Inspiration is a simple bedside method to stress the right ventricle. This increases the venous return to the heart. A normal healthy right ventricle can cope up with this increased inflow facilitated by the reduced pulmonary vascular resistance during inspiration. A failing right ventricle will not be able to cope up with this increased inflow. This is reflected as an increased right ventricular end diastolic pressure which is transmitted to the right atrium and hence to the jugular vein. As a result mean JVP increases during inspiration, which is called as Kussmaul's sign.

Pulsus paradoxus - with extensive damage to the right ventricle associated with inferior wall infarction, pulsus paradoxus

has been reported. This has to be differentiated from pericardial effusion and cardiac tamponade.

Constrictive pericarditis, restrictive cardiomyopathy, cardiac tamponade and pulmonary embolism should be considered in the differential diagnosis of right ventricular infarction.

Clinical triad

Distended neck veins

Clear lung fields

Hypotension

Electrocardiographic changes

Standard 12 lead ECG plus right ventricular lead and posterior chest leads, (18 lead ECG) are always necessary to diagnose right ventricular myocardial infarction.

ST segment elevation greater than 1mm in right chest leads, especially V_4R is essential to diagnose right ventricular myocardial infarction^{56,64-71}.

Presence of Qs complex in V_4R , $ST V_2 / ST aVF$ ratio less than 0.5, ST elevation in V_1 with diminishing ST elevation in V_2 and V_3 are also supposed to indicate right ventricular myocardial infarction.

Failure of reciprocal ST segment depression to develop in the right precordial leads in the case of inferior wall infarction is also significant.

Lewis⁷⁰ and associates reported the hyperacute phase of right ventricular myocardial infarction. This is manifested as slope elevation of ST segment in leads V₁ and V₄R or only in lead V₄R.

Occlusion of right coronary artery, proximal to right ventricular branch is diagnosed by changes in V₄R.

ST segment elevation V₄R with a positive 'T' wave predicts proximal right coronary artery occlusion.

Absence of ST elevation with positive 'T' wave predicts a distal right coronary artery occlusion.

Presence of ST segment depression in anterior leads points to right coronary artery occlusion.

Markers used in the Diagnosis of Acute Myocardial Infarction

S. No	Marker	Time of initial elevation (Hrs)	Mean time to elevation (Hrs)	Time to return to normal range	Most common sampling schedule
1	Troponin-I	3-12	24	5-10 days	Once atleast 12 hrs after chest pain
2	Troponin-T	3-12	12-48	5-14 days	Once atleast 12 hrs after chest pain
3	CK-MB	3-12	24	2-3 days	Every 12 hrs x 3 days
4	Myoglobin	1-4	6-7	24 hours	Frequently 1-2 hour chest pain
5	LDH	10	24-48	10-14 days	Once atleast 12 hrs after chest pain

Enzymatic criteria for diagnosis of Myocardial Infarction⁷²

- ❖ 15 % of CK in the myocardium is CK- MB, which provides sensitivity and specificity for its diagnostic marker of acute myocardial infarction
- ❖ Serial increase, then decrease of plasma CK-MB, with a change > 25 % between any 2 values
- ❖ CK-MB > 10-13 u/L or more than 5% of total CK
- ❖ CK-MB / CK activity more than 2.5 indicates myocardial rather than skeletal muscle as a source of CK-MB

- ❖ Increase in CK-MB activity > 50% between any 2 samples, separated by atleast 4 hours
- ❖ If only a single sample available, CK-MB elevation > 2 fold
- ❖ Beyond 72 hours, an elevation of Troponin T or I or LDH1>LDH 2

Early diagnosis of myocardial infarction is offered by CK-MB subforms and Myoglobin of which CK-MB subforms are most specific.

- ❖ Absolute level of CK-MB 2 isoform greater than 1.0 u/l within 4-6 hrs of myocardial infarction. (OR)
- ❖ CK- MB 2/ CK- MB 1 greater than 2.5 has sensitivity & specificity around 90%⁷³

Troponins

- ❖ Cardiac specific troponins which are highly sensitive and specific marker
- ❖ They increase after Myocardial Infarction to the level over 20 times higher than the cut-off value
- ❖ Levels of Troponin-I may remain elevated for 7-10 days and Troponin-T may remain elevated for upto 10-14 days.

Myoglobin

- ❖ First cardiac marker that rises above normal range after acute myocardial infarction
- ❖ Lacks cardiac specificity and levels return to normal range within 24 hours of the onset of MI.

Diagnosis of re-infarction⁷⁴

- ❖ Serum cardiac marker that remains elevated in the blood briefly such as CK-MB or Myoglobin is useful.
- ❖ Reinfarction defined as the secondary elevation of CK-MB activity, 36-48 hrs after onset symptoms and the value should be more than 50% above the preceding baseline.

Other Biochemical alterations

- ❖ Serum cholesterol and lipoprotein fractions are relatively unchanged in the initial 1-2 days but decrease significantly over subsequent days and weeks. Hence lipid measurements are done in 24-48 hours or 6-8 weeks later⁷⁵.
- ❖ White blood cell count show mild to moderate elevation in 3-5 days.
- ❖ ESR raises slowly and remains elevated for 1-2 weeks.

Imaging Techniques

Chest Roentgenogram

- ❖ The chest film is used to exclude other causes of chest pain such as pneumothorax, pulmonary infarction with effusion, aortic dissection, skeletal fracture and so on.
- ❖ It is also used to evaluate cardiac size, the presence of pulmonary edema and atherosclerotic changes⁷⁶.

Echocardiography

- ❖ Early detection of the presence or absence of wall motion abnormalities can aid in the management.
- ❖ Estimation of left ventricular function by echocardiogram is useful prognostically⁷⁷.
- ❖ Useful in the diagnosis of right ventricular infarction, ventricular aneurysm, pericardial effusion and left ventricular thrombus⁷⁸. Doppler echocardiography is useful in the diagnosis of ventricular septal defect and acute mitral regurgitation.

Radionuclide Assay

❖ Technetium 99m sestamibi scan

It has potential use in identifying infarct in patients with atypical presentations or in patients with ECGs that are not interpretable. Normal findings are associated with extremely low risk of subsequent cardiac events.

❖ Thallium scanning⁷⁹

Thallium accumulates in the viable myocardium.

- Perfusion imaging has been used in risk stratification after myocardial infarction and for measurement of infarct size to evaluate reperfusion therapies.
 - Recent advances include dual-score 64-slice CT scanning that can do a full scan in 10 seconds and produce high resolution images that allow fine details of the patient's coronary arteries to be seen. This technology allows for noninvasive and early diagnosis of coronary artery disease and thus earlier treatment before the coronary arteries become more or completely occluded
- ❖ MRI can identify wall thinning scar, delayed enhancement (infarction), and wall motion abnormalities (ischemia).

Currently, this is not a primary diagnostic modality for MI, but coronary artery assessment may be enhanced by magnetic resonance angiography (MRA) in the future.

MANAGEMENT STRATEGIES

Prehospital care

The overall goal is to reduce mortality and morbidity of acute Myocardial Infarction. A means to end this is to reduce the time interval from the onset of symptoms to the provision of resuscitation skills, adequate analgesia, adequate assessment and diagnosis and where appropriate, early thrombolytic therapy^{80,81}.

Strategies

- ❖ Educating patients regarding recognition of symptoms and call for help.
- ❖ Role of paramedics in recognizing and treating life threatening arrhythmias.
- ❖ Integrated response by general practitioners, ambulance service and hospital staff is required.
- ❖ Prehospital thrombolysis - Alteplase for this purpose.

Initial Management

- ❖ Initiation of intravenous line and administration of 5 % Dexrose / saline to keep the vein opened
- ❖ Continuous monitoring of cardiac rhythm, heart rate, blood pressure and other vital signs.

Oxygen administration

Hypoxemia is common due to ventilation perfusion mismatch. Oxygen administration has been reported to decrease ST-segment elevation in anterior myocardial infarction. So supplementation of Oxygen (2-4 L/min) for all causes of uncomplicated myocardial infarction with SaO₂ greater than 90% is justified for 2-3 hours. Oxygen therapy should be extended for patients with pulmonary congestion and desaturation⁸³.

Analgesia

- ❖ The pain and anxiety contribute to the over activity of autonomic nervous system which increases the myocardial oxygen demand.
- ❖ Morphine is the drug of choice. It is given at a dose of 2-4 mg intravenously at intervals of 5-15 minutes, until pain is relieved.

- ❖ Caution is needed in inferior infarct complicated by right ventricular myocardial infarction⁸⁴, as it produces severe bradycardia due to vagotonic effect.
- ❖ Morphine allays anxiety, relieves pain, causes vasodilation and therefore reduces preload.
- ❖ Pethidine is preferable in patients with inferior wall and right ventricular myocardial infarction.

Nitrates (Sublingual)

- ❖ Decreases myocardial oxygen demand and increases myocardial oxygen supply. (dilate infarct related vessels)
- ❖ Sublingual nitroglycerine up to 0.4 mg, 3 doses can be administered at about 5 minutes intervals⁸⁴.
- ❖ Should be avoided in the right ventricular myocardial infarction.

Beta blockers

- ❖ Relieves pain by diminishing myocardial oxygen demand.
- ❖ Reduces frequency of progression of threatened infarction to complete infarction. It also reduces the infarct size.
- ❖ Reduces life threatening arrhythmias
- ❖ Should be initiated within first 3 hours of infarction.

Metoprolol is used in the dose of 5 mg intravenously every 2-5 minutes for total of 3 doses. 15 minutes after the last intravenous dose, oral dose of 50 mg every 6 hours for 48 hours, then 100 mg every 12 hours⁸² is given.

Aspirin

The use of non enteric coated chewable Aspirin 160-325 mg taken at the onset of symptoms causes 25 % decrease in the mortality. It rapidly inhibits cyclo-oxygenase irreversibly in platelets, followed by reduction of Thromboxane A2 formation, a mediator of platelet aggregation⁸⁴.

In patients with aspirin allergy or contraindication, Clopidogrel in the dose of 300 mg to 600 mg stratum dose is recommended.

Recent data from the CLARITY TIMI 28 (Clopidogrel as Adjunctive Reperfusion Therapy - Thrombolysis in Myocardial Infarction [TIMI] 28)⁸⁵ suggest that adding Clopidogrel to this Aspirin and thrombolysis regimen is safe and effective. The Clopidogrel Dose used was 300 mg. Further studies suggest that a higher dose of Clopidogrel may have added benefit.

Reperfusion in Myocardial Infarction

When ST segment elevation of atleast 2mm in two contiguous precordial leads and 1mm in two adjacent limb leads is present, patient should be considered for reperfusion therapy⁸⁴

Primary Percutaneous coronary intervention

- ❖ Primary Percutaneous coronary intervention is effective in restoring perfusion in STEMI when carried out on an emergency basis in the first few hours of MI⁸⁴.
- ❖ It is more effective in restoring patency of occluded coronary arteries better short term and long term clinical outcomes.
- ❖ Primary PCI is preferred when the diagnosis is in doubt, cardiogenic shock is present, bleeding risk is increased or symptoms have been present for atleast 2-3 hours when the clot is more mature and less easily lysed by fibrinolytic agents.

Thrombolysis

Thrombolytic therapy reduces the relative risk of in hospital death by 50% when given in the first hour of onset of symptoms.

Other uses

- ❖ Limits infarct size

- ❖ Limits LV dysfunction
- ❖ Reduce the incidence of serious complications as septal rupture, cardiogenic shock & malignant arrhythmias

Benefit of thrombolysis possible up to 12 hours, especially if chest discomfort is still present and ST segment remain elevated in ECG. Goal of thrombolysis is to achieve TIMI-grade 3, flow in the infarct related artery.

Drugs used in thrombolysis

Drug	Dose
Streptokinase	1.5 million units over period of 1 hour intravenously
Recombinant tissue Plasminogen activator (rt-PA)	Initial bolus of 15 mg, followed by an infusion of 50 mg or 0.75 mg per kg body weight over next 30 minutes and 30 mg or 0.5 mg/kg over subsequent 60 minutes
Reteplase (r-PA)	Initial bolus of 15 Mega units followed by a second bolus of 15 Mega units in 30 minutes
Tenecteplase (TNK-tPA)	Direct 0.5 mg/kg bolus

Unless contra indicated, thrombolytic therapy should be given ideally within 3 hours, preferably within 6 hours and can be justifiably given up to 12 hours.

Rt-PA is more effective than streptokinase in restoring the perfusion (TIMI- grade 3flow). As bolus fibrinolysis has the advantage of ease of administration, a lower chance of medication error and less non cerebral bleeding, bolus fibrinolytics like reteplase or tenecteplase can be preferred over t-PA.

Criteria for thrombolysis

- ❖ Chest pain consistent with angina
- ❖ ECG changes
 - ST ↑ greater than 1 mm in 2 or more contiguous limb leads
 - ST ↑ greater than 2mm in 2 or more contiguous precordial leads
 - New onset LBBB
- ❖ Absence of contraindications

Alternative reperfusion regimens⁸³

- Combined intravenous glycoprotein II b /IIIa inhibitor with a reduced dose of fibrinolytic agent

- Facilitated PCI : Glycoprotein II b /IIIa inhibitors followed by PCI
- Pharmacoinvasive approach : Pharmacologic reperfusion regimen followed by routine angiography and PCI after a delay of 12 - 24 hours
- Rescue PCI if failure of reperfusion beyond 90 minutes of thrombolysis
- Elective PCI in coronary artery re-occlusion or recurrent ischemia.

Antithrombotic agents

Drugs

1. Unfractionated heparin

Immediate administration of Unfractionated heparin helps to facilitate thrombolysis and establish the patency of infarct related artery in t-PA regimen of thrombolysis. It also gives mortality benefit when added to aspirin and streptokinase regimen.

Dose:

- Bolus 60 u/kg
- Maintenance of 12 U/ Kg / hr
- To keep aPTT – 1.5 to 2 times the control value

2. Low molecular weight heparins

They have several advantages over unfractionated heparin due to increased anti-factor Xa : IIa ratio, decreased sensitivity to platelet factor IV, as more stable and reliable anticoagulant effect and enhanced bio-availability. There is no need for aPTT monitoring.

Drugs used

Nadroparin

Deltaparin

} Efficacy similar to unfractionated heparin

Enoxaparin - superior to unfractionated heparin

(enoxaparin 1 mg/kg subcutaneously every 12 hours)

3. Platelet Glycoprotein IIb/IIIa receptor inhibitors

These drugs facilitate thrombolysis and reduce the rate of reocclusion of reperfused vessels. They act by inhibiting the platelet glycoprotein IIb/IIIa receptors of platelets surface which are the final common pathway in platelet aggregation.

Drugs:

- Abciximab is a monoclonal antibody to the IIb/IIIa receptor. It is given in the dose of 0.25 mg/kg intravenous bolus followed by 10 mcg/min over 12 hours.
- Orbofiban, sibrafiban
- Tirofiban
- Eptifibatide
- These drugs are more useful in NSTEMI

4. Bivalirudin (a direct thrombin inhibitor) has shown some promise in the setting of STEMI if combined with high-dose clopidogrel load and may be an appropriate alternative strategy.

Other drugs

1. **ACE inhibitors** : These should be prescribed within 24 hours of AMI to all patients with myocardial infarction and overt congestive cardiac failure. It reduces the ventricular remodelling after infarction thereby reduces the subsequent development of failure
2. **Statins** : Statin therapy should be initiated prior to hospital discharge, and perhaps as early as possible, in all patients with an STEMI. High dose Atorvastatin 80 mg/day used in the PROVE IT-TIMI 22 and MIRACL trials⁸⁶ was found effective.

Special efforts in the management of Right Ventricular Myocardial Infarction

The importance of diagnosing right ventricular myocardial infarction lies in its management. The drugs routinely used in treatment of myocardial infarction like sedatives, analgesics, anticoagulants, antiplatelet drugs, thrombolytics etc can be given.

However because of the unique hemodynamic features of right ventricular myocardial infarction the use of diuretics and nitrates may precipitate an impending or worsen a preexisting low output state.

Volume Expansion

Since there is an imbalance between right and left ventricular filling pressures volume expansion has been advocated to improve the low output state in right ventricular infarction⁸⁷.

Volume expansion should be continued until hypotension is corrected (or) till the pulmonary capillary wedge pressure reaches 15-20 mm of Hg (or) till right atrial pressure reaches the level of atleast 14-15 mm of Hg.

If central hemodynamic monitoring is not available, one to two liters of saline can be infused while closely following the blood pressure and urine output and examining the patient for signs of

pulmonary congestion. If volume loading does not improve hemodynamics or is not tolerated, inotropic therapy should be tried.

But if the left ventricle is also extensively damaged volume expansion will be ineffective. Right atrial pacing has been advocated as an alternative to volume loading.

Drugs

Inotropic agents are indicated when hypoperfusion persists despite adequate left ventricular filling pressure. Dopamine and Dobutamine may increase the cardiac index and ejection fraction⁸⁷.

When left ventricular failure is present arterial vasodilators are added to this regimen. They decrease the impedance to left ventricular outflow. Consequently left ventricular diastolic, left atrial and pulmonary arterial pressure also decrease.

This lowers the impedance to right ventricular outflow and enhance right ventricular output. Intra aortic balloon counter pulsation has been used in a few occasions. But it is not clear whether it is useful in a patient with dominant right ventricular infarction.

Management of Bradyarrhythmias:

Bradyarrhythmias can be corrected by vagolytics. Temporary pacing may be necessary in refractory cases. When pacing is done, atrioventricular sequential pacing is done as this improves right sided forward flow. Replacement of tricuspid valve has been carried out in treatment of severe tricuspid regurgitation secondary to right ventricular myocardial infarction⁸⁸.

Complications of Myocardial Infarction

Mechanical Complications

- Ventricular dysfunction
- Cardiogenic shock
- Mitral regurgitation
- Ventricular septal rupture
- Free wall rupture – (cardiorrhesis)

Electrical Complications

Tachyarrhythmias

- These include ventricular premature beats, ventricular tachycardia, ventricular fibrillation and accelerated idioventricular rhythm.

- Supraventricular arrhythmias—sinus tachycardia is the common one. Other common arrhythmias are atrial fibrillation, atrial flutter and paroxysmal supraventricular tachycardia.
- Accelerated junctional rhythm is more common in inferoposterior myocardial infarction

Bradyarrhythmias

- Sinus bradycardia is more common with inferoposterior & right ventricular myocardial infarction managed by atropine. Persistent bradycardia may need pacing.
- AV blocks are particularly common with inferior infarct, attributed to ischemia and enhanced vagal activity and the release of adenosine
- First degree block needs no specific management
- Second degree block particularly high grade type II blocks may require pacing.
- Complete heart blocks are common in inferior infarct and they are transient and get corrected with reperfusion.
- Intra ventricular conduction abnormalities of which LBBB is more common.

Specific Complications of Right Ventricular Myocardial Infarction

1. Hypotension
2. Conduction defects
3. Thromboembolic phenomenon with a special predilection for pulmonary embolism
4. Severe tricuspid insufficiency
5. Pericarditis
6. Chronic right heart failure in survivors⁸⁸
7. Rupture of Ventricular free wall and interventricular septum⁸⁹
8. Right to left shunting through a patent foramen ovale^{90,91}
9. Right ventricular aneurysm⁹²

MATERIALS AND METHODS

The study was conducted in the Intensive Coronary Care Unit of Government Kilpauk Medical College Hospital, Chennai. The study period was from February 2009 to October 2009.

Design of the study: Prospective Study

Ethical clearance: Obtained

Inclusion Criteria:

- All patients admitted in Intensive Coronary Care Unit with ST elevation in V4R were taken for study
- Both male and female patients were included
- Both young and old patients were included

Exclusion Criteria:

- Patients with other congenital and acquired valvular heart disease, COPD and Right ventricular hypertrophy.
- Patients with previous myocardial infarction
- Patients with other serious comorbid illness

Study Protocol

1. Clinical

2. Electrocardiogram

Clinical Study

In each case detailed history about age, sex, occupation, presenting symptoms, time of admission, previous history and habits were taken.

Comprehensive risk factor analysis was made with specific importance to

- Obesity
- Diabetes Mellitus
- Hypertension
- Hyperlipidemia
- Smoking status- classified into
 - ❖ Non smoker
 - ❖ Ex smoker if discontinued 10 years ago and
 - ❖ Current smokers
- Family H/o premature coronary artery disease, defined as sudden cardiac death, acute myocardial infarction, coronary artery disease before the age of 40 years.

Detailed Physical Examination was Made Including

- Vital signs
- Blood pressure
- JVP

- Kussmaul sign
- Cardiac examination for gallops, murmur, rub, rales
- Clinical evidence of tricuspid regurgitation
- Risk stratification with 'Killips' classification

All patients of acute myocardial infarction were given the following treatment.

- Absolute bed rest
- Intravenous line – 5% dextrose to keep the vein open
- Nasal O₂ by mask or prongs
- Aspirin 325 mg tablet chewed & swallowed
- Narcotic pain reliever – Inj. pethidine 50 mg intramuscularly
- Beta blockers, Nitrates and Diuretics are usually avoided as they may cause Hypotension
- Enalapril was started 6 hrs after beta blockers if there was no hypotension
- Sedatives and stool softeners were routinely administered
- Intravenous fluid – Normal Saline was given if hypotension was present. Initially Normal saline was given at the rate of 100 ml/hr. If hypotension was not controlled with volume

expansion inotropic agents like dobutamine and dopamine were added to this regimen.

- Anticoagulant - Unfractionated heparin was started 6 hrs after thrombolysis - dose 5000 u i.v. qid

Thrombolysis

Thrombolysis was done depending on the merits of the patient and therapeutic window, excluding contraindications. Thrombolysis was done with Streptokinase 1.5 Million units dissolved in 100 ml of Normal Saline administered over a period of 1 hour after cardiac monitoring. Adequacy of thrombolysis was assessed in 90 minutes after thrombolysis based on the following parameters.

1. Control of chest pain
2. ST segment resolution atleast 50%
3. Reperfusion arrhythmias

Patients were continuously monitored for complications like infarct extension, re-infarction, left ventricular dysfunction, cardiogenic shock, mitral regurgitation and ventricular septal rupture and managed accordingly. Electrical complications were continuously monitored and prompt measures were done to correct the same.

Complete basic investigations were done in all patients,

- Urine - albumin, sugar, deposits.
- Urine ketones (if diabetic)
- Blood - TC, DC, Hb%, ESR.
- Blood - Glucose, Urea, Creatinine
- Serum Electrolytes
- X ray Chest PA view
- Serial CPK-MB estimation was done on admission and every 12 hours for first 48 hours.
- Lipid profile was done in all patients on the day of admission

Echocardiography

All patients were subjected to echocardiography after stabilization or at discharge.

The following parameters were noted.

- Wall motion abnormalities in the form of :
 - ❖ Hypokinesia – reduced systolic movement
 - ❖ Akinesia - absence of systolic movement
 - ❖ Dyskinesia - paradoxical motion

- Presence of aneurysm, mitral regurgitation, left ventricular clot or pericardial effusion were noted. Ejection fraction was determined by M-Mode and 2-D using Simpson's rule.
- This was done for patients benefit, but this data was not included in our study.

The patient outcome at the end of first week was assessed in relation to mortality, morbidity with respect to in-hospital complications like electrical, mechanical, left ventricular dysfunction, mitral regurgitation, etc.

OBSERVATION & ANALYSIS

Study period: February 2009 – October 2009

Prospective Study

Total of 53 patients were studied

Clinical Observation

1. Incidence

Acute Myocardial Infarction : 283

Acute Inferior wall Myocardial Infarction : 148

Acute Right Ventricular Myocardial Infarction : 53

All the 53 cases were associated with Inferior wall myocardial infarction.

2. Sex

Out of 53 cases, 40 cases were males and 13 cases were females.

Table 1
Sex Prevalance

Sex	Number	Percentage (%)
Male	40	75.47
Female	13	24.53
Toal	53	100

3. Age

Ages of the patients taken for the study ranging from 35 to 80 years.

- 19 patients were aged between 50-59 years
- The youngest patient in this study was 36 years
- Oldest patient was 80 years old
- In our study highest incidence was in the 6th decade

Table 2

Age Prevalance

Age	Male	Male (%)	Female	Female (%)	Total	Total (%)
30-39	5	9.44	1	1.89	6	11.32
40-49	9	16.98	2	3.77	11	20.75
50-59	15	28.3	4	7.55	19	35.85
60-69	8	15.09	3	5.66	11	20.75
70-79	3	5.66	2	3.77	5	9.44
80-89	-	-	1	1.89	1	1.89
Total	40	75.47	13	24.53	53	100

4. Occupation

As the occupation of the patient were one of the risk factor, detailed occupational history was analysed in all the patients in this study.

Table 3**Occupation**

Occupation	No. of Patients	Percentage (%)
Professional	4	7.55
Teachers, Clerical staff, Sedentary workers	11	20.75
House wives	13	24.53
Labourers, Coolies	25	47.17
Total	53	100%

5. Admission time interval after the onset of initial symptom**Table 4****Admission time intervals after onset of chestpain**

Time window	Male	Female	Total	Percentage (%)
<3 hour	14	2	16	30.19
3-6 hour	14	6	20	37.74
6-12 hour	11	4	15	28.30
12-24 hour	1	1	2	3.77

6. Diurnal variation of initial symptom

The day was divided into 4 quarters

- Maximum number of patients (19) had their initial symptom in the second quarter between 6A.M.and12P.M.

- 17 patients had their initial symptoms between 12 A.M. to 6 A.M.

Table 5

Diurnal variation of Initial symptoms

Time	Male	Female	Total	Total (%)
12 A.M. to 6.00 A.M.	13	4	17	32.07
6 A.M. to 12 P.M.	14	5	19	35.85
12 P.M. to 6 P.M.	11	2	13	24.53
6 P.M. to 12 A.M.	2	2	4	7.55

7. Analysis of Clinical features (Presenting Symptom)

- Angina was present in 71.7% of patient
- Angina equivalent in 28.3 %

Table 6

Analysis of presenting Symptoms

Presenting Symptom	Male	Female	Total	Total %
Angina	30	8	38	71.7
Angina Equivalent	10	5	15	28.3

- All patients with chest pain were associated with sweating
- Nausea, Vomiting and Indigestion were the common presenting angina equivalents in young
- Dyspnoea was the predominant symptom in elderly people
- Most of the patients with angina equivalents were Diabetes

Table 7

Analysis of presenting symptoms

Gender	Angina Equivalent	Dysnoea	Syncope	Giddiness	Nausea/ Vomiting	Palpitation	Indigestion
M	10	11	4	8	9	5	3
F	5	2	2	2	3	1	2
Total	15	13	6	10	12	6	5

8. Risk Factors Analysis

Risk factors like Diabetes Mellitus, Hypertension, Smoking, Obesity, Family history and Hypercholesterolemia were taken for analysis.

- 14 patients had Diabetes Mellitus
- 19 patients were hypertensive
- 26 patients were smokers
- 18 patients had obesity
- 4 patients presented with family h/o Coronary artery Heart Disease

Table 8
Analysis of Risk factors

Factor	Male	Female	Total	Total %
Diabetes Mellitus	11	3	14	26.4
Hypertension	15	4	19	36
Smoking	26	-	26	49.06
Obesity	14	4	28	34
Family History	3	1	4	7.5

9. Clinical Signs Suggestive of RVMI.

Table 9
Analysis of Clinical features

No	Clinical signs	No. of Cases	Total %
1	Hypotension SBP < 90	26	49.06
2	Bradycardia	34	64.25
3	Elevated JVP	16	30.19
4	Kussmaul's Sign	10	18.87
5	Basal Rales	16	30.19
7	Cardiogenic shock	7	13.2

10. Risk stratification by Killip Classification

Table 10

Killip Classification

Killip class	No of Cases	Total %	Death
1	37	69.81	
2	7	13.21	
3	5	9.43	
4	4	7.55	3

Electrocardiographic Observations

1. ECG Analysis for ST elevation

Table 11

ECG analysis

ST elevation in V4R	No of patients	Percentage (%)	No of Death
1mm	46	86.79	1
>1mm	7	13.21	2
Total	53	100	3

2. Arrhythmias

- 43.34% patients developed I degree heart block
- 20.75% patients developed II degree heart block

- 13.2% patients developed complete heart block
- 1.89% patients developed Atrial Fibrillation
- 5.66% patients developed Ventricular Tachycardia

Table 12

Analysis of Arrhythmias

Type	No of cases	Percentage (%)
I degree heart block	23	43.34
II degree heart block	11	20.75
Complete heart block	7	13.20
RBBB	2	3.77
LAHB	2	3.77
AF	1	1.89
VT/VF	3	5.66

Clinical course of the patients after thrombolysis

1. Improvement of clinical symptoms after thrombolysis

- Chestpain, syncope, nausea and vomiting improved dramatically with thrombolysis
- 53.85% patients with dyspnoea were also improved with thrombolysis

Table 13**Improvement of Clinical Symptoms After Thrombolysis**

S.No	Presenting Symptom	No of patients	Symptoms improved in no of patients after thrombolysis	Total (%)
1	Chestpain	38	34	89.47
2	Nausea/vomiting	12	12	100
3	Syncope	6	6	100
4	Dyspnoea	13	7	53.85

2. Age related mortality ratio

- Out of 53 cases 3 patients died

Table 14**Age related mortality ratio**

Age group	No of cases	Death	Percentage of Death
30-39	6	-	-
40-49	11	-	-
50-59	19	1	5.2
60-69	11	1	9.09
70-79	5	1	20
80-89	1	-	-
Total	53	3	5.66

DISCUSSION

A steady decline in the mortality rate from acute myocardial infarction has been observed since 1970. In recent decades, thrombolytic therapy has dramatically reduced the mortality from acute myocardial infarction.

Right ventricular myocardial infarction is usually associated with inferior wall myocardial infarction, with occurrence of many complications. So, during early period of right ventricular myocardial infarction we have to carefully watch for the early complications.

The value of conventional ECG as a simple non invasive bedside method for diagnosing right ventricular myocardial infarction has increased recently⁴. Many cases of right ventricular myocardial infarction which were missed during bedside clinical examination were identified by electrocardiography.

A. Clinical Analysis

Age and Sex Distribution

In this study males were affected more than females. This observation usually correlates well with many studies regarding acute right ventricular myocardial infarction³⁻¹⁰. This may be due to the risk factors like hypertension, smoking, alcoholism and type A personality which were common in males than females.

Age Distribution

Most of the patients affected were aged between 50-59 years. But in the western studies the highest incidence was in the 7th and 8th decade⁹⁴

In this study most of the females affected were in the older age group than males. This may be due to the protective hormonal effect offered during the premenopausal period⁹⁴.

Occupation

Various other studies showed that the greater incidence was among those who did sedentary work like executive, businessmen and other professionals, perhaps some of the rich people may prefer the private sector. But as this study was conducted in Government hospital, greater incidence was noted among the labourers, coolies and weavers.

In this study women after menopause, had increased incidence of myocardial infarction, this is seen especially among house wives who lead a sedentary life.

Admission time interval and prognosis

Three deaths reported in this study occurred in patients who got delayed admission. This delay might be due to poor transport facilities

in the village areas particularly at night and also due to atypical chest pain.

Inference :

This explains the necessity of integrated health services in the management of acute myocardial infarction and the need for the early transport of the patients to the coronary care units.

Diurnal variation

Maximum number of patients reported their initial symptom in the second quarter of the day between 6 A.M. and 12 P.M. It revealed a pronounced circadian periodicity for the time of onset of the acute myocardial infarction with the peak incidence of events between 6 A.M. and 12 P.M. It is supported by **MILIS⁹⁵ study**.

Reason :

This is due to the early morning rise in the plasma catecholamine and cortisol.

Presenting Symptoms

In right ventricular myocardial infarction nausea with or without vomiting was the presenting symptom irrespective of angina. Angina equivalents were more common in females. It was around 30%. According to Harrison's Principles of Internal Medicine a

minimum 15-20% of Myocardial Infarction are painless⁸³. The silent infarcts are greater in patients with diabetes.

Presenting symptoms observed did not vary from various other published studies. As with previous studies prevalence of angina equivalents was 28% and a higher proportion has been reported in patients with diabetes. Similar observation were made in other studies⁹⁶.

Nausea and vomiting were found to be a common association as given in published observations.

Reasons :

In myocardial infarction stimulation of nerve fiber in an ischemic zone of myocardium surrounding the necrotic central area probably give rise to pain. Diabetics with autonomic neuropathy have blunting of this response.

Inferior wall infarction frequently causes increased diaphragmatic and vagal activity which causes vomiting / nausea more frequently.

Risk factors

Garg et al⁹⁷ and Croft et al⁹⁸ observed Diabetes, hypertension and smoking as major risk factors for Myocardial infarction. This study findings are also consistent with them.

36% of patients admitted with inferior wall and right ventricular myocardial infarction were found to be hypertensive, equal incidence in males and females. Smoking was exclusively prevalent among males with a frequency of 49 %. Central abdominal obesity, a particular risk factor in India, was found in 34% of patients with myocardial infarction, more often with females.

Reasons :

The sedentary life style of Indian females, diet habits and lesser inclination to accept modern ways of life styles which involves maintenance of body make up could have contributed this finding.

Inferences :

The clinicians should now concentrate more at identifying those patients who are at increased risk of acute myocardial infarction and benefit from more aggressive prophylactic cardiovascular treatment to prevent it from occurring. More attention should be paid towards prediabetics and prehypertensives to prevent the acute myocardial infarction.

Presenting signs

Hypotension with bradycardia was present in 60 % of patients, a similar clinical observation in other studies. They were transient in many patients. This is due to the activity of Bezold Jarish reflex.

Cohn³ and **Cintron**⁹⁹ reported 70-100% incidence of hypotension. **Croft**⁹⁸ et al and **Garg et al**⁹⁷ reported 33.8% and 8% incidence of hypotension in patients with and without right ventricular myocardial infarction.

So, whenever a patient with inferior wall myocardial infarction presents with hypotension a co-existent right ventricular myocardial infarction should be suspected.

Systemic venous congestion and raised jugular venous pressure were present in 30% of the patients. And also a significant number of patients had Kussmaul's sign, oliguria and right ventricular S₃.

The features of right ventricular failure noted in right ventricular myocardial infarction patients in this study are marginally lower than those reported by **Cohn**³ et al.

Cintron⁹⁹ reported incidence of right ventricular failure almost similar to that observed in this study.

However in the studies by **wacker**¹⁰ et al and **Braat**¹⁰⁰ et al none of the patients with right ventricular myocardial infarction, had clinical features of right ventricular failure. So the clinical features of right ventricular failure need not be present in all cases and are present only in cases with severe right ventricular involvement.

Though hypotension with clear lung fields suggestive of right ventricular myocardial infarction, significant number of patients with

right ventricular myocardial infarction had basal rates. This probably was due to the coexistent left ventricular inferior wall myocardial infarction in all these patients.

B. Electrocardiographic analysis

Gupta¹¹ et al and **Morgera¹³ et al** proposed ST elevation more than 0.1 mV in the right precordial leads especially in V4R is 100% specific for the diagnosis of right ventricular myocardial infarction. Based on that observation, in this study only those who had ST elevation in V4R were taken for observations.

Braat et al¹⁰⁰ and Kleinho et al in circulation 1983 reported that ST elevation in right precordial leads is transient. However in this study ST elevation was persistent upto 48 hours after initial ECG in more than 10 % of patients.

This was similar to **Jha¹⁴ et al** and **croft et al⁹⁸** who reported that ST elevation may persist for longer duration upto 72 hours.

ST elevation in right precordial leads indicates right ventricular myocardial damage exceeding 25% or damage reaching the lateral margins of the right ventricle. So, ST elevation in V4R also indicates extent of the right ventricular damage.

In this study, the lesser the magnitude of ST elevation, lesser the complication was noted. Death rate was observed in patients who had

ST elevation more than 1 mm. This shows that the severity of the infarction can be assessed by the amount of ST elevation.

The serious complications like cardiogenic shock, arrhythmias were observed in patients with ST elevation more than 1mm in this study. This correlates well with other studies⁹⁹ also.

Bradycardia: Bradyarrhythmias are the commonest electrical complication noted in this study. This is due to stimulation of cardiac vagal afferent receptors.

In this study bradycardia was noted in 2/3 of patients. **Braat et al**¹⁰⁰ reported that the incidence of sinus bradycardia in patients with and without right ventricular infarction to be 30 % and 10 % respectively.

In this study also a statistically significant higher incidence of bradycardia was observed. Most of the patients improved with thrombolytic therapy. Similar observation were made by **Cintron**⁹⁹ et al.

Conduction Defects: Victor Legrand⁴⁵ reported 42% incidence of atrioventricular conduction disturbances in his patients with right ventricular myocardial infarction while **K. C. Garg**⁹⁷ et al reported an incidence of 36.4 %.

In this study the incidence of atrio-ventricular conduction disturbances was 77.36%. Looking into specific conduction

disturbances a lower incidence of complete atrio-ventricular block was observed in this study.

Of the 7 cases with complete heart block, 2 patients expired during hospital stay.

Atrial Fibrillation which was transient noticed in 1 patient (1.89 %). This might be due to left ventricular dysfunction or atrial ischemia. Reported incidence of atrial fibrillation in AMI is 10-15%, but it is only one third in those with IWMI, as compared to anterior wall MI¹⁰¹.

The incidence of intraventricular conduction defects in this study was 7.5%, i.e., 2 cases had complete RBBB and another 2 cases had left anterior hemi block. The reported incidence of RBBB in AMI is 2-4% with pure IWMI and 7-10% with RV extension and that of LAHB is 3.5% in various studies¹⁰⁰.

C. Clinical course of the patients Cardinal Symptoms of Acute Myocardial infarction like angina, nausea, vomiting and breathlessness were improved after thrombolysis.

Early thrombolytic therapy improves the survival rate in patients with acute myocardial infarction. After thrombolysis there was improvement in patients with hypotension and cardiogenic shock. Out of 53 patients 3 patients died which is comparable with other studies⁹⁹.

CONCLUSION

1. The incidence of Right Ventricular Myocardial Infarction is fairly common (35.8%) and its more common in males than females and the difference being less as the age advances.
2. In majority of patients retrosternal chest pain and anginal equivalents like syncope had its onset between 6A.M. and 12P.M.
3. Smoking and Hypertension were the most prevalent risk factors.
4. All cases of **Inferior Wall Myocardial Infarction should have Right sided chest leads recorded during ECG examination** as more the ST elevation more the severity of Right Ventricular Myocardial Infarction and its complications and mortality rate.
5. If diagnosis of Right Ventricular Myocardial Infarction is correctly made earlier and thrombolysed the prognosis is usually good even in patients with complications.
6. Mortality is higher in patients with Inferior wall Myocardial Infarction with Right Ventricular Myocardial Infarction because of higher incidence of conduction disturbances and pump failure.

SUMMARY

Right Ventricular Myocardial Infarction is usually associated with Inferior wall Myocardial Infarction, which carries higher mortality and morbidity when it is associated with Inferior wall Myocardial Infarction. Conventional ECG with 12 leads will miss Right Ventricular Myocardial Infarction. So 18 lead ECG is essential to pick up Right Ventricular Myocardial Infarction and Posterior wall Myocardial Infarction early.

Early identification and immediate thrombolysis will improve the clinical course and prognosis of Right Ventricular Myocardial Infarction and also reduces the mortality and morbidity many fold.

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ABBREVIATIONS

AMI	–	Acute myocardial infarction
AV	–	Atrioventricular
CK	–	Creatine Kinase
DC	–	Differential count
DM	–	Diabetes Mellitus
ECG	–	Electro-Cardiogram
ESR	–	Erythrocyte sedimentation rate
Hb	–	Hemoglobin
HDL	–	High density lipoprotein
ICCU	–	Intensive coronary care unit
IWMI	–	Inferior Wall Myocardial Infarction
JVP	–	Juglar Venous Pressure
LBBS	–	Left Bundle Branch Block
LDH	–	Lactate dehydrogenase
LDL	–	Low density lipoprotein
MI	–	Myocardial Infarction
PCI	–	Percutaneous Coronary Intervention
RV	–	Right Ventricle.
RVMI	–	Right Ventricular Myocardial Infarction
TC	–	Total count
t -PA	–	Tissue Plasminogen Activator.

PROFORMA

A Study on Clinical Profile of RVTI

Name :

Age :

Sex :

Occupation :

Address :

Date of admission :

Time of admission :

Presenting Symptoms

1. Chest pain : Y/N

Time of Onset

2. Angina Equivalents

Dyspnoea Syncope Sweating Palpitation Giddiness

Indigestion

Past History :

- DM
- HT
- IHD
- CVA
- PVD
- Dyslipidemia

Personal History

Diet	Veg / Non Veg
------	---------------

Smoking	Y / N
---------	-------

Alcohol Y / N

Menstrual History

Oral contraceptives	Y / N
---------------------	-------

Family History

DM

HT

IHD

CVA

Dyslipidemia

General Examination:

General Appearance : Comfortable
Anxious
Apprehensive

Extremities : Warm
Cold
Sweating

Anemia : Y/N

Cyanosis : Y/N

Clubbing : Y/N

Pedal edema : Y/N

Vital Signs:

Pulse :

Blood Pressure :

Respiratory Rate :

Temperature :

System Examination

Cardio Vasular System

- JVP :
- Kussmaul sign :
- Heart sounds : S1 S2 Normal / muffled
- Murmur :
- Pericardial rub :

Respiratory System

- Vesicular Breath Sounds -
- Crepitations
- Basal -
- Upto mid axilla -
- Upto one half -
- Extensive -
- Rhonchi -
- Others -

Abdomen

- Bowel sounds Y / N
- Hepatomegaly Y / N
- Splenomegaly Y / N
- Free fluid/ ascites Y / N

Central Nervous System

- Level of sensorium :
- Any focal neurological deficit :

Investigations

1. Urine - Albumin
Sugar
Deposits

2. Blood - Hb
TC
DC
ESR

3. Blood - Urea
Sugar
Creatinine
Electrolytes

4. Lipid Profile
Total cholesterol
Triglycerides
LDL
HDL

5. Serum CK
LDH
SGOT
SGPT

6. Chest X-ray

7. Electrocardiogram

15 lead ECG -

Analysis of ECG

- Rate
- Rhythm
- ST elevation in
- ST elevation in V4R mm
- Reciprocal ST depression
- Heart Blocks

Repeat ECG after 90 min of Thrombolysis

Course in the hospital:

Improved - Transferred to Ward

If death - Cause of death

No. days stayed in ICCU :

Outcome:

CHART – 1
SEX PREVALANCE

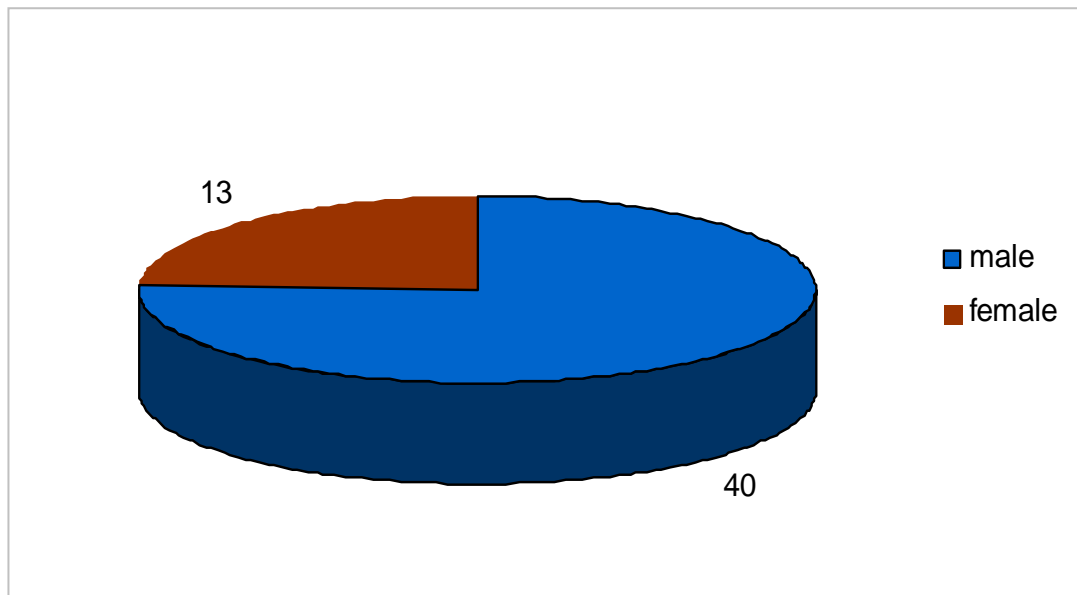


CHART – 2
AGE PREVALANCE

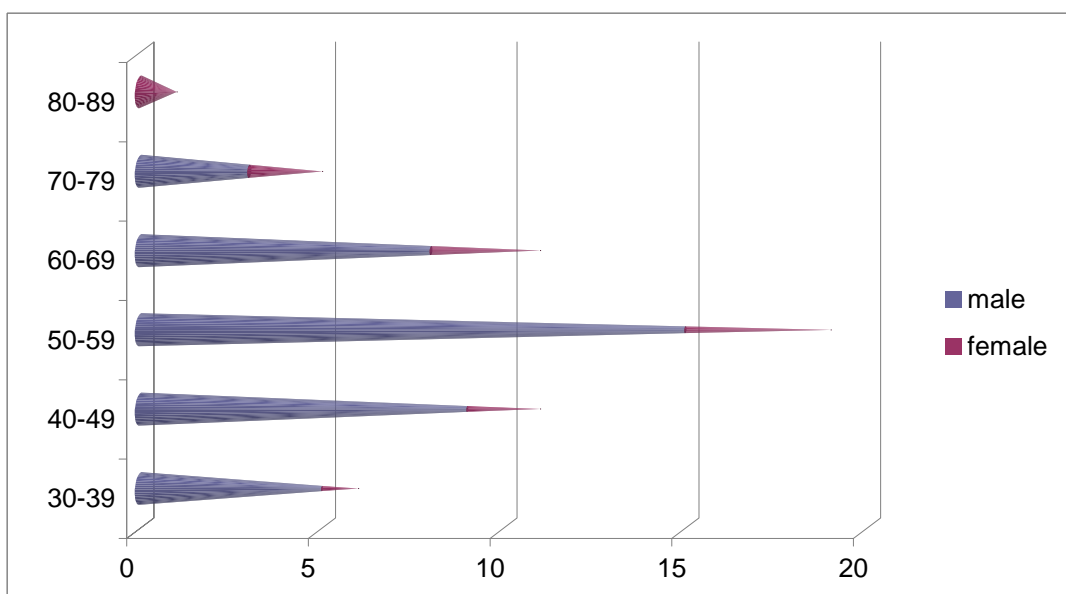


CHART – 3

DIURNAL VARIATION OF SYMPTOMS

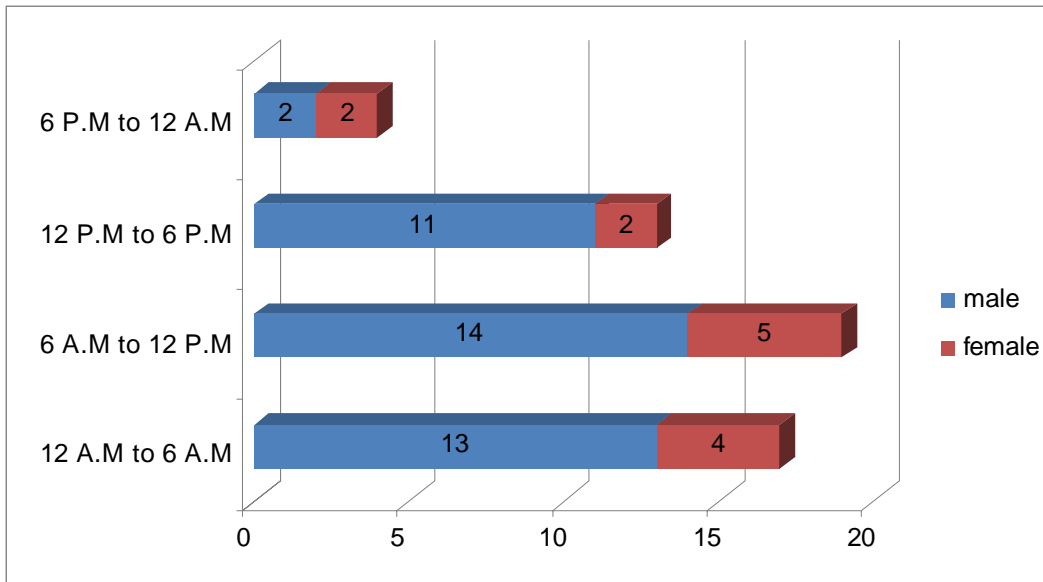
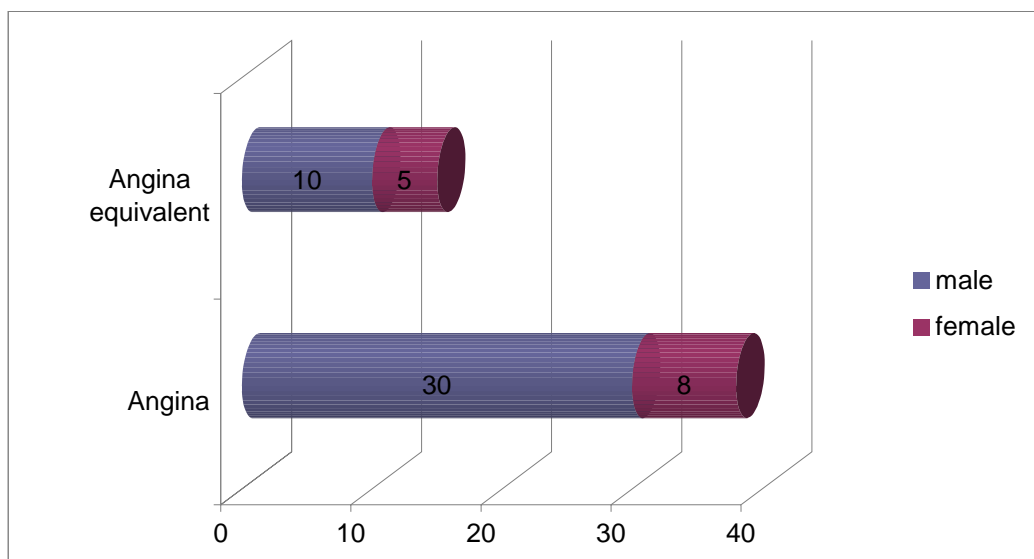
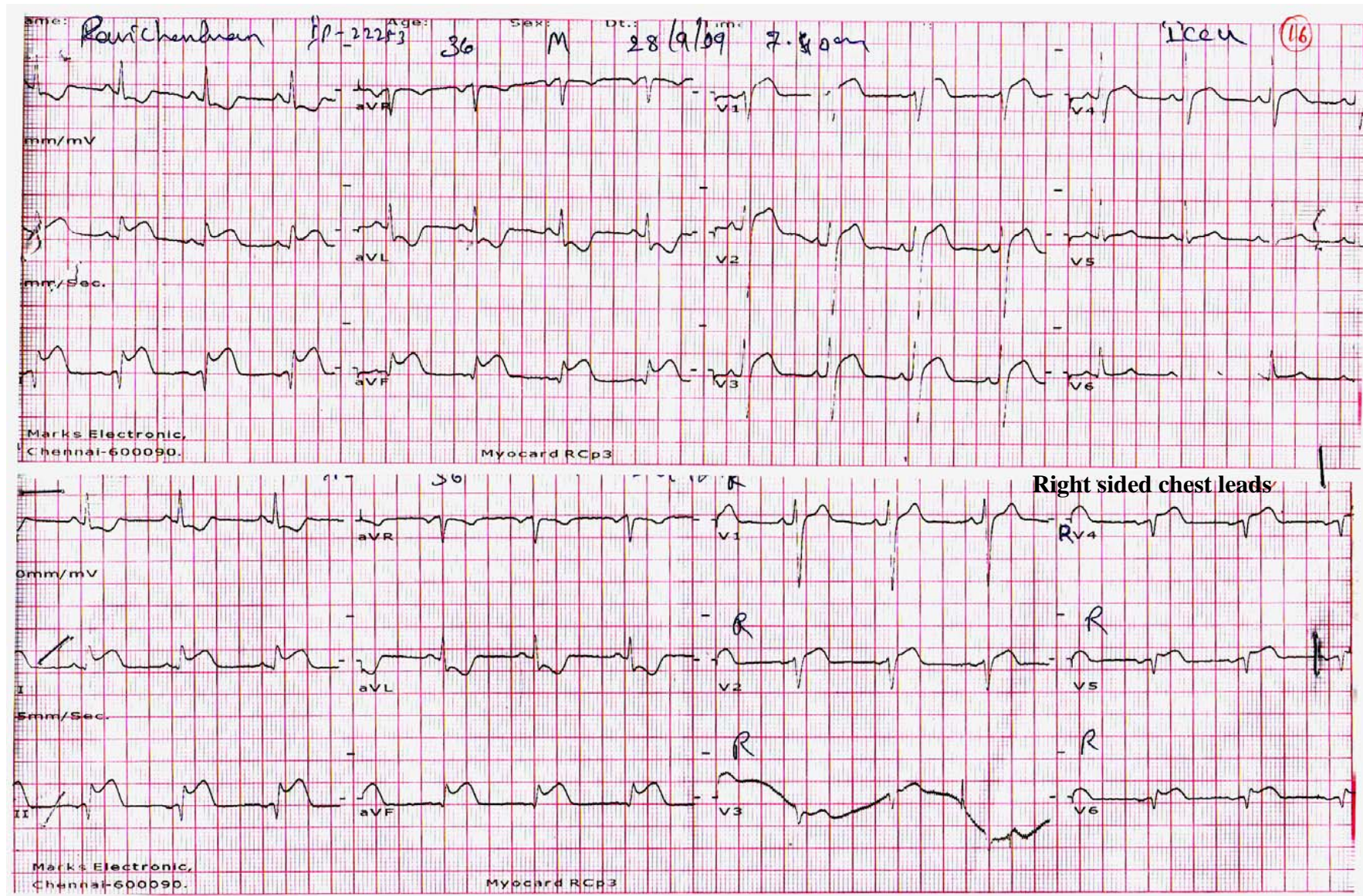


CHART – 4

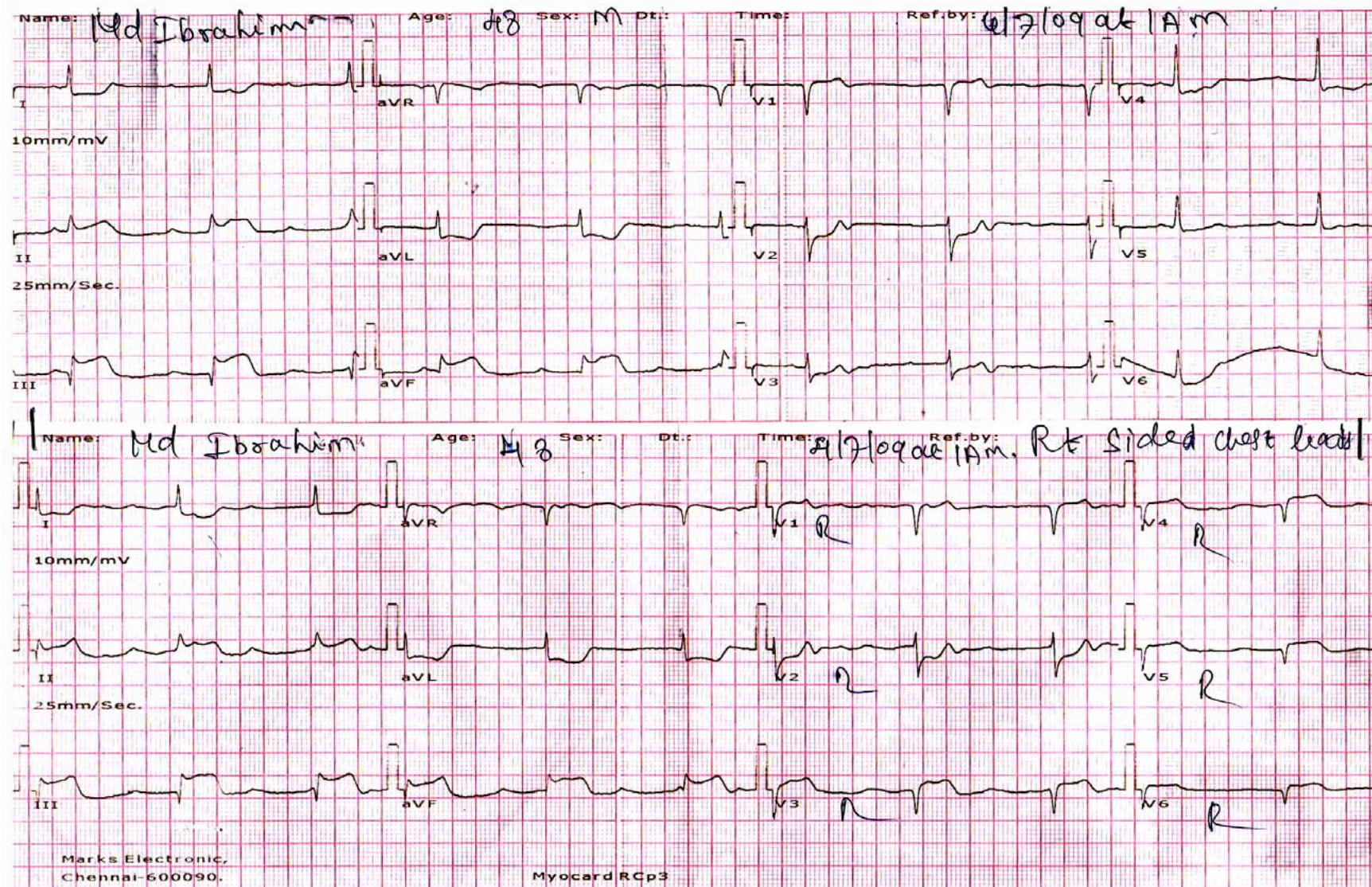
PRESENTING SYMPTOM



ACUTE IWMI WITH RVMI WITH NO CONDUCTION DISTURBANCES



ACUTE IWMI WITH RVMI WITH AV CONDUCTION DISTURBANCE



MASTER CHART

S.No	Age	Sex	Occupation	Angina	Anginal eq	Pain duration	DM	HT	Smoking	Obesity	FHO CAHD	Bradycardia	Hypotension	JVP	Basal rates	Killip	ST elevation mm	1 av block	2 av block	3 av block	RBBB/LAHBB	AF/VT/VF	Death
1	50	F	HW	A		4		+							+	1	1	+					
2	50	M	LAB		+	1	+	+	+			+	+		+	2	1	+			LAFB		
3	57	M	SW		+	8	+	+	+			+	+	+	+	2	1	+					
4	50	F	HW	A		6				+		+				1	1		+				
5	39	M	LAB	A		6			+							1	1						
6	55	M	LAB	A		3		+	+							1	1	+					
7	80	F	HW		+	6	+			+		+	+	+	+	2	1	+					
8	36	M	P	A		2			+			+				1	1	+					
9	50	M	LAB	A		5			+			+	+	+	+	4	2			+		VT	
10	65	M	SW		+	3	+		+	+		+	+	+	+	3	2.5	+			RBBB		
11	39	F	HW		+	3	+				+	+	+		+	2	1					VT	
12	62	M	SW	A		4						+	+	+	+	2	1			+			
13	40	M	LAB		+	1	+	+	+							1	1						
14	40	M	LAB	A		6			+							1	1	+					
15	68	M	LAB	A		1			+			+	+	+	+	3	3	+				VT	
16	60	M	SW	A		2				+		+	+			1	1				RBBB		
17	53	M	LAB	A		4			+							1	1		+				
18	52	M	LAB	A		2			+			+				1	1				LAFB		
19	40	M	LAB	A		2	+	+		+						1	1						
20	48	M	P	A		1	+		+			+	+			1	1		+	+			
21	55	M	LAB	A		7				+						1	1	+					
22	38	M	P	A		2			+	+		+				1	1						
23	50	M	LAB	A		6		+				+	+	+		4	1		+				+
24	56	M	LAB	A		4		+	+		+					1	1	+					
25	64	M	SW		+	5						+	+			1	1						
26	61	M	LAB	A		8			+			+	+	+		4	3			+		AF	+
27	70	F	HW		+	7						+	+	+		4	2			+			+
28	62	F	HW	A		4						+				1	1						

29	70	M	SW		+	6	+					+	+			1	1	+					
30	58	M	LAB	A		5			+	+		+				1	1		+				
31	70	F	HW		+	3		+				+	+	+		3	2		+				
32	47	M	LAB	A		7	+	+	+							1	1						
33	60	F	HW	A		10				+						1	1		+				
34	64	F	HW	A		11		+				+	+	+		1	1		+				
35	48	M	LAB	A		9		+		+						1	1	+					
36	74	M	SW		+	6		+				+	+	+		3	2	+					
37	50	M	LAB	A		8	+		+	+		+	+			1	1			+			
38	37	M	LAB	A		12			+	+	+	+				1	1	+					
39	58	F	HW	A		8						+	+			1	1	+					
40	61	M	SW	A		16		+				+				1	1		+				
41	49	F	HW		+	20	+									1	1						
42	46	M	LAB	A		2			+			+	+			1	1	+					
43	54	M	lab	A		7		+		+			+	+		2	1	+					
44	66	M	SW	A		2			+	+						1	1	+					
45	56	M	LAB	A		10		+		+		+	+	+		2	1			+			
46	75	M	SW		+	4		+					+	+		3	1		+				
47	40	M	P	A		1	+		+			+	+			1	1		+				
48	55	M	LAB		+	6		+		+						1	1	+					
49	55	F	HW	A		4		+								1	1	+					
50	36	M	SW		+	12	+		+		+	+				1	1						
51	50	M	LAB	A		5			+			+				1	1	+					
52	49	F	HW	A		5				+		+	+	+		1	1	+					
53	45	M	LAB	A		9			+	+						1	1						

KEY

P - Professional
SW - Sedentary Worker
LAB - Labourer
HW - House Wife
A - Angina
DM - Diabetes Mellitus

HT - Hypertension
FHO - Family History of
VT - Ventricular tachycardia
VF - Ventricular Fibrillation
AF - Atrial Fibrillation
RBBB - Right Bundle Branch Block
LAFB - Left Anterior Fascicular Block